Hypohydration and thermoregulation in cold air

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O'Brien, Catherine, Andrew J. Young, and Michael N. Sawka. Hypohydration and thermoregulation in cold air. *J. Appl. Physiol.* 84(1): 185–189, 1998.—This study examined the effects of hypohydration on thermoregulation during cold exposure. In addition, the independent influences of hypohydration-associated hypertonicity and hypovolemia were investigated. Nine male volunteers were monitored for 30 min at 25°C, then for 120 min at 7°C, under three counterbalanced conditions: euhydration (Eu), hypertonic hypohydration (HH), and isotonic hypohydration (IH). Hypohydration was achieved 12 h before cold exposure by inducing sweating (HH) or by ingestion of furosemide (IH). Body weight decrease (4.1 ± 0.2%) caused by hypohydration was similar for HH and IH, but differences ($P < 0.05$) were found between HH and IH in plasma osmolality (292 ± 1 vs. 284 ± 1 mosmol/kgH$_2$O) and plasma volume reduction (−8 ± 2 vs. −18 ± 3%). Heat debt (349 ± 14 among) did not differ ($P > 0.05$) among trials. Mean skin temperature decreased throughout cold exposure during Eu but plateaued after 90 min during HH and IH. Forearm-finger temperature gradient tended ($P = 0.06$) to be greater during Eu (10.0 ± 0.7°C) than during HH or IH (8.9 ± 0.7°C). This suggests weaker vasoconstrictor tone during hypohydration than during Eu. Final mean skin temperature was higher for HH than for Eu or IH (23.5 ± 0.3, 22.6 ± 0.4, and 22.9 ± 0.3°C, respectively), and insulation was lower on HH than on IH (0.13 ± 0.01 vs. 0.15 ± 0.01°C·W^{-1}·m^{-2}, respectively), but not with Eu (0.14 ± 0.01°C·W^{-1}·m^{-2}). This provides some evidence that hypertonicity impairs the vasoconstrictor response to cold. Although mild hypohydration did not affect body heat balance during 2-h whole body exposure to moderate cold, hypohydration-associated hypertonicity may have subtle effects on vasoconstriction that could become important during a more severe cold exposure.

hypovolemia; hypertonicity; furosemide; cold-induced vasodilation

COLD EXPOSURE elicits physiological responses that minimize heat loss (vasoconstriction) and increase heat production (shivering). These thermoregulatory effector responses are modulated via the hypothalamic thermoregulatory centers and are based on afferent input from both skin temperatures (T_{sk}) and core temperatures (2). Hypohydration [reduced total body water (TBW)] alters central and peripheral thermoregulatory controls during heat stress (12, 16), but whether this effect also occurs during cold stress is unknown. Although the medical literature suggests that hypohydration is a predisposing factor for hypothermia (5), this possibility has not been evaluated in controlled experiments. Cold exposure also elicits cold-induced vasodilation (CIVD), periodic increases in extremity blood flow that are thought to prevent excessive tissue cooling and injury (11). Some evidence suggests that hypohydration impairs the CIVD response, an effect that could increase susceptibility to peripheral cold injury (14, 15).

During cold-weather outdoor activities, individuals often become hypohydrated by 3–8% of their body weight (6). Reasons for this fluid deficit include high sweat losses, blunted thirst, cold-induced diuresis, increased respiratory water losses, conscious underdrinking, and poor availability of water (6). Most of these factors cause greater water than solute losses, eliciting a hypertonic hypohydration (HH) (3). However, cold-induced diuresis causes proportional water and solute losses, eliciting an isotonic hypohydration (IH) (10). During heat stress, hypertonicity and hypovolemia have independent effects on thermoregulatory effector responses (sweating and skin blood flow) (16). Hypertonicity acts centrally on osmoreceptors in the hypothalamus to affect sweat rate, whereas hypovolemia acts on cardiopulmonary baroreceptors to alter skin blood flow (16). Whether hypertonicity and hypovolemia exert independent effects on thermoregulation during cold exposure is unknown.

This study investigated the effects of hypohydration (4–5% body weight loss) on body temperatures, metabolic responses, and body-heat balance during cold exposure. Our experimental approach allowed isolation of potential effects of hypertonicity and hypovolemia on thermoregulatory responses. We hypothesized that hypovolemia would reduce blood flow to the periphery and blunt the CIVD response and that the central actions of hypertonicity would impair shivering thermogenesis and vasoconstriction.

METHODS

Subjects. Nine men gave their voluntary and informed consent to participate in these experiments, which were approved by the appropriate Institutional Review Boards. Volunteers were medically cleared before participating in this study. Preliminary body-composition analysis was conducted to exclude subjects whose body fat exceeded 20%. Subjects characteristics were: age, 24 ± 2 (mean ± SE) yr; height, 178 ± 2 cm; weight, 77 ± 4 kg; TBW, 49 ± 3 liters; body fat, 15 ± 1%; body surface area, 1.94 ± 0.06 m^2; and peak oxygen uptake ($\dot{V}O_{2peak}$), 55 ± 1 ml·kg^{-1}·min^{-1}. Investigators adhered to AR 70–25 and US Army Medical Research and Materiel Command Regulation 70–25 on the Use of Volunteers in Research.

Protocol. For each subject, the study measurements were completed over a span of ~5 wk, including a 10-day preliminary period and three experimental trials, each spaced 1 wk apart. The order in which the experimental trials were completed [one euhydration (Eu) and two HH] was counterbalanced. Subject participation spanned the period from July to January. Because each subject completed all experimental testing within 15 days, served as his own control, and worked indoors when not being tested, any seasonal difference that may have occurred was not expected to affect the results.

During the preliminary period, subjects measured and recorded their nude body weight each morning after voiding and before breakfast. Averaging these weights established

the body weight that represented euhydration for each subject. During the preliminary period, body composition, $\dot{V}O_{2peak}$, and TBW were also measured. Body density (D) was determined by hydrostatic weighing, with simultaneous residual volume measurements by nitrogen washout (8). Body composition (%fat) was calculated from D by using Siri's equation [$(4.95/D - 4.5) \cdot 100$] (20). $\dot{V}O_{2peak}$ was determined to characterize the fitness level of the subjects. An incremental treadmill-running protocol was used (12). TBW was determined by stable-isotope dilution (7). Briefly, each subject was given a 30-g dose of deuterium oxide (^2H_2O) followed by 100 ml tap water. Blood samples were obtained immediately before isotope ingestion and 3 h after ingestion. Serum was purified in diffusion dishes, and the concentration of ^2H_2O was analyzed on an infrared spectrophotometer.

Each experimental trial consisted of 30-min rest in temperate (25°C) ambient air, followed by 120-min rest in cold air [7°C, 40% relative humidity (RH)]. Subjects arrived at 0700, after fasting overnight. They were weighed, and, over the next 60 min, they were instrumented with an esophageal thermocouple, intravenous catheter, skin thermocouples, and electrocardiogram (ECG) electrodes. The esophageal thermocouple was placed at heart level (estimated as $0.25 \cdot$ height) (18). Skin thermocouples were placed at nine sites on the right side of the body. Metabolic rate ($\dot{M}$) was measured by open-circuit spirometry. Blood samples were obtained from an indwelling catheter placed into an antecubital vein and maintained patent with heparinized saline. Heart rate was obtained from ECG telemetry. Blood pressure was measured by auscultation with a sphygmomanometer.

After being instrumented, the subjects (dressed in shorts, socks, and shoes) rested semisupine on a nylon-mesh lounge chair for the 30-min preexposure at 25°C. Subjects lay on wool blankets and were comfortable in this nearly thermoneutral environment. Body temperatures were recorded every minute, and heart rate was recorded every 5 min. $\dot{M}$ was measured after 20 min. A blood sample was taken after 25 min, followed by measurement of blood pressure. The subject then stood, voided his bladder, and entered the climatic chamber. In the climatic chamber (7°C, 40% RH), subjects again reclined, semisupine, on a nylon-mesh lounge chair. Body temperatures and heart rate were continually measured. $\dot{M}$ was measured continuously for the first 30 min and then for 10 min during each 30-min period thereafter. Blood pressure was measured every 30 min. A blood sample was drawn after 60 min and again after 120 min. At the end of the 120-min exposure, the subject stood, voided his bladder, and exited the climatic chamber. Cold exposure was terminated sooner if the subject's core temperature fell to 35°C or if he requested to be removed from the chamber.

The experimental trials were completed at the same time of day on three separate occasions. On one occasion, subjects were tested when euhydrated (normal food and fluid intake the day before; nothing to eat or drink the morning of the testing). On the other two occasions, subjects were tested when hypohydrated by 4–5% of their baseline body weight. Two methods of dehydration were employed to induce HH on one occasion and IH on the other. HH was achieved by using a standardized exercise-heat protocol (12) the day before the HH trial. Briefly, the subject reported to the laboratory and performed 3–4 h of intermittent light-intensity exercise in the heat (40°C, 20% RH) to induce sweating. Throughout exercise, the fluid replacement was restricted. Water loss through sweating results in increased solute in the plasma, which then exerts an osmotic gradient to redistribute water to the plasma (17); thus hypovolemia is limited, while tonicity increases. IH was achieved by administration of a diuretic

(furosemide) on the day before the IH trial. Furosemide inhibits renal reabsorption of sodium and chloride, thereby reducing water reabsorption by the kidney and increasing urine formation. The loss of both solute and water results in hypovolemia with minimal change in tonicity. Subjects were given 40 mg of furosemide at 0930, 1530, and, if sufficient weight loss had not been achieved, at 2030. Weight loss was accomplished via urinary losses combined with restricted fluid intake. For HH and IH, subjects were given a light supper, with restricted fluid intake after achieving the target body weight.

Experimental procedures. Body temperatures were measured and recorded every minute. Mean weighted T_{sk} ($\overline{T}_{sk}$) was calculated from the formula: $\overline{T}_{sk} = 0.175\, T_{chest} + 0.07\, T_{upperarm} + 0.07\, T_{forehead} + 0.175\, T_{back} + 0.05\, T_{hand} + 0.07$ forearm temperature (T_{fa}) $+ 0.19\, T_{thigh} + 0.20\, T_{calf}$ (see Ref. 13). Core-$\overline{T}_{sk}$ gradient was calculated as the difference between esophageal temperature (T_{es}) and $\overline{T}_{sk}$ ($T_{es} - \overline{T}_{sk}$). T_{fa}-finger temperature gradient was calculated as the difference between T_{fa} and middle-finger temperature (T_f), or $T_{fa} - T_f$. The following indexes of CIVD were determined from records of T_f: initial minimum temperature achieved during cold exposure, time to reach that temperature, and number of CIVD, which were defined as a temperature difference between a local minimum and maximum of at least 0.5°C.

$\dot{M}$ was calculated from oxygen uptake ($\dot{V}O_2$) and the caloric equivalent of the respiratory exchange ratio. Insulation was calculated as ($T_{es} - \overline{T}_{sk}$)/$\dot{M}$. Heat debt was determined by partitional calorimetry (S) and thermometric (HD) methods (21) at 5-min intervals for the first 30 min in the cold and every 30 min thereafter. The calorimetric method calculated the difference between heat production and heat loss, according to the formula: $S = \dot{M} - (W + L + E + K) - (R + C)$, expressed in W/m². The work rate (W) was 0 in this experiment; respiratory heat loss by convection and evaporation (L) was 8% of $\dot{M}$; heat loss by evaporation (E) was 4.1 W/m² in the cold; heat loss by conduction (K) was 0 in this experiment; and dry-heat losses by radiation and convection (R + C) was $10.2 \cdot (\overline{T}_{sk} - T_{db})$, where T_{db} is the dry bulb temperature. Mean body temperature ($\overline{T}_b$) was calculated from $\overline{T}_b = \chi T_{es} + (1 - \chi)\overline{T}_{sk}$, where χ is the weighting coefficient for thermoneutral (0.79) or cold (0.67) (see Ref. 1). The thermometric method calculated heat debt by the formula $HD = \Delta\overline{T}_b \cdot mass \cdot$ specific heat of body tissues ($3.47\,kJ \cdot kg^{-1} \cdot °C^{-1}$).

Blood samples were obtained without stasis and with control of posture and arm position. Blood samples were analyzed for hematocrit, hemoglobin concentration, osmolality, and glucose. Plasma volume was predicted for Eu trials (19). Calculations for the percent change in plasma volume (from Eu to hypohydration and from pre- to cold exposure) were made from the corresponding changes in hemoglobin and hematocrit values (4). Plasma volume was calculated for all other conditions from the appropriate predicted and percent change values. Urine volume was used to calculate urine flow for both baseline and cold exposure.

Statistical analyses. Data were analyzed by using commercial software (Statistica, StatSoft). A two-factor (time and trial) analysis of variance for repeated measures was performed. The statistical significance level was set at $P < 0.05$. Tukey's honestly significant difference post hoc test was applied when main effects or interactions were found to be statistically significant. All data are reported as means ± SE.

RESULTS

Ambient conditions during both the preexposure baseline period [$T_{db} = 24.9 \pm 0.2$°C; dew point tempera-

ture (T_{dp}) = 11.4 ± 1.1°C] and cold exposure (T_{db} = 6.9 ± 0.02°C; T_{dp} = −5.2 ± 0.3°C, equivalent to 41.6% RH) did not differ across trials. Only one subject did not complete all the trials; he was withdrawn after 105 min on Eu because of a fall in core temperature that reached the medical safety limit. All measurements except $\dot{M}$ were obtained just before that subject left the chamber; these measurements were used in the data analysis.

Hydration and fluids. Body weight, plasma volume, and plasma osmolality are shown in Table 1. Body weight loss was similar ($P > 0.05$) for HH (4.9 ± 0.2%) and IH (4.3 ± 0.2%). Plasma volume decreased ($P < 0.05$) and plasma osmolality increased ($P < 0.05$) during the subsequent cold exposure in all trials. Plasma glucose values remained >4.0 mM on all occasions; i.e., no subject was hypoglycemic before or during any trial. Urine flow rate increased ($P < 0.05$) from baseline to cold exposure during Eu (from 0.76 ± 0.17 to 2.70 ± 0.54 ml/min) but not during HH (from 0.27 ± 0.05 to 0.55 ± 0.04 ml/min) or IH (from 0.28 ± 0.08 to 0.45 ± 0.12 ml/min).

Thermal responses. T_{es} is shown in Fig. 1. Although there was no main effect between trials, an interaction effect indicated that T_{es} began to plateau after 60-min cold exposure during HH. During both Eu and IH, however, T_{es} continued to fall throughout the cold exposure ($P < 0.05$). There were no differences ($P > 0.05$) in $\overline{T}_{sk}$ between trials (Fig. 2). The large fall in $\overline{T}_{sk}$ on cold exposure increases the variability of the data; therefore, $\overline{T}_{sk}$ data were also statistically analyzed without including preexposure values. This analysis revealed a significant interaction, showing that $\overline{T}_{sk}$ continued to fall ($P < 0.05$) throughout Eu cold exposure, whereas $\overline{T}_{sk}$ began to plateau after 90 min during both hypohydration trials. Core temperature-$\overline{T}_{sk}$ gradient increased ($P < 0.05$) from baseline throughout cold exposure similarly ($P > 0.05$) for Eu (from 3.9 ± 0.2 to 13.7 ± 0.3°C), HH (from 3.7 ± 0.2 to 13.0 ± 0.3°C), and IH (from 4.4 ± 0.2 to 13.4 ± 0.3°C). Again, the analysis of cold-exposure values without including preexposure

Fig. 1. Effect of cold exposure on esophageal temperature (T_{es}) (mean ± SE) during euhydration (Eu, ○), hypertonic hypohydration (HH, ■), and isotonic hypohydration (IH, ▲) trials. There was no main effect between trials ($P > 0.05$). *Interaction effect showed a plateau during HH after 60 min, whereas T_{es} continued to fall during both Eu and IH; $P < 0.05$.

values revealed that T_{es}-$\overline{T}_{sk}$ gradient began to plateau after 90 min during both hypohydration trials, whereas it continued to increase ($P < 0.05$) during Eu. T_{fa}-T_f gradient increased from preexposure (1.75 ± 0.46°C) to cold exposure (8.99 ± 0.44°C at 120 min), but it did not differ ($P > 0.05$) between trials.

$\overline{T}_f$ during cold exposure (10.4 ± 0.2°C) did not differ between trials ($P > 0.05$). Two subjects demonstrated no CIVD response during any trial. Of the remaining seven subjects, five exhibited CIVD during Eu, seven during HH, and four during IH. Only two subjects demonstrated CIVD during all three trials. There was no difference between trials ($P > 0.05$) for the initial minimum temperature (9.1 ± 0.2°C), or the time to

Table 1. *Body weight, plasma volume, and plasma osmolality*

Trial	Baseline	Cold Exposure
Body weight, kg		
Eu	76.5 ± 3.7	
HH	73.1 ± 3.6*	
IH	73.6 ± 3.7*	
Plasma volume, liters		
Eu	3.3 ± 0.1	2.8 ± 0.1
HH	3.1 ± 0.1	2.7 ± 0.1
IH	2.7 ± 0.2*	2.3 ± 0.2*
Plasma osmolality, mmol/kgH₂O		
Eu	280 ± 1	283 ± 2
HH	292 ± 1*	293 ± 2*
IH	284 ± 1*	288 ± 3

Values are means ± SE. Baseline (preexposure) and cold exposure (120 min) values are shown for euhydration (Eu), hypertonic hypohydration (HH), and isotonic hypohydration (IH) trials. *Significant difference from Eu ($P < 0.05$).
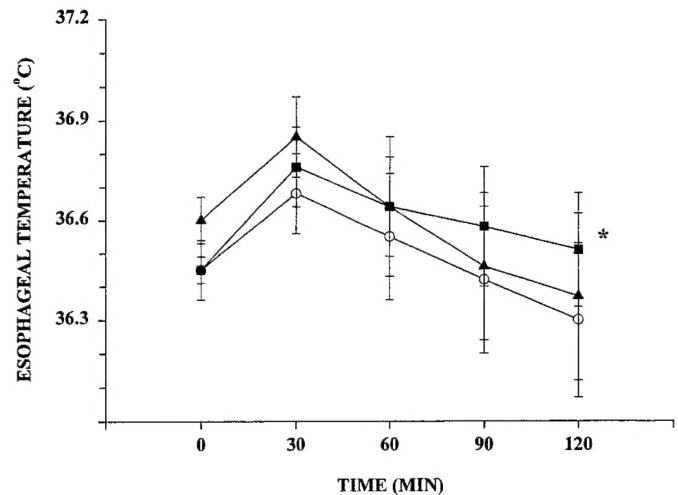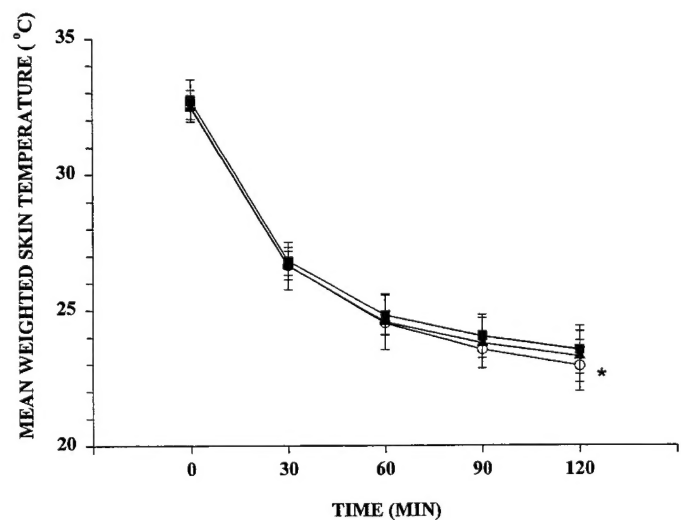

Fig. 2. Effect of cold exposure on mean skin temperature ($\overline{T}_{sk}$) (mean ± SE) during Eu (○), HH (■), and IH (▲) trials. There was no main effect between trials ($P > 0.05$). When only cold-exposure values were analyzed, an interaction effect showed a plateau during both hypohydration trials after 90 min. *$\overline{T}_{sk}$ continued to decrease during Eu; $P < 0.05$.

reach that temperature (55 ± 3 min), or the initial rate of fall in temperature (0.30 ± 0.01°C/min).

M increased ($P < 0.05$) from baseline (36.5 ± 1.4 W/m^2) throughout cold exposure (113.6 ± 4.2 W/m^2), with no differences ($P > 0.05$) between trials. Insulation, shown in Fig. 3, was higher ($P < 0.05$) in IH (0.15 ± 0.01°C·W^{-1}·m^{-2}) than in HH (0.13 ± 0.01°C·W^{-1}·m^{-2}) but not in Eu (0.14 ± 0.01°C·W^{-1}·m^{-2}). Figure 4 provides heat-debt analyses performed by using both calorimetric and thermometric calculations. Heat debt calculated either way increased similarly ($P > 0.05$) on all trials.

Cardiovascular responses. Heart rate increased ($P < 0.05$) during the first 30 min of cold exposure but did not change thereafter. Heart rate was higher ($P < 0.05$) during IH [from 64 ± 2 beats/min (bpm) preexposure to 71 ± 4 bpm at 120-min cold exposure] than Eu (58 ± 2 to 66 ± 4 bpm) but not HH (58 ± 2 to 71 ± 4 bpm). Systolic blood pressure increased ($P < 0.05$) from baseline to 120 min of cold exposure (104.2 ± 1.5 to 124.8 ± 2.6 mmHg), but there were no differences between trials. Diastolic blood pressure (70.4 ± 1.8 to 77.2 ± 2.1 mmHg), mean arterial pressure (81.7 ± 1.5 to 93.1 ± 2.1 mmHg), and pulse pressure (33.8 ± 3.0 to 47.6 ± 1.9 mmHg) all increased ($P < 0.05$) with cold exposure but were similar ($P > 0.05$) between trials.

DISCUSSION

This study investigated the effects of hypohydration on thermoregulation during cold exposure. By using two different methods of dehydration, the independent influences of hypovolemia and hypertonicity were also examined. It was anticipated that hypovolemia would reduce peripheral skin blood flow, thereby resulting in lower peripheral T_{sk} and blunted CIVD. The central actions of hypertonicity were expected to impair shivering thermogenesis and maintenance of vasoconstrictor tone, leading to warmer T_{sk} at the expense of greater
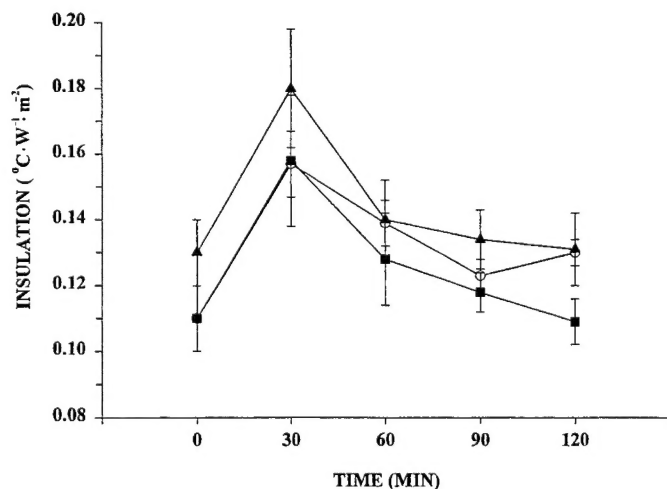

Fig. 4. Effect of cold exposure on heat debt (mean ±SE) on Eu (○), HH (■), and IH (▲) trials. There were no significant differences ($P > 0.05$) between trials for either thermometric (solid lines) or calorimetric (dashed lines) calculations of heat debt.

Fig. 3. Effect of cold exposure on insulation (mean ± SE) during Eu (○), HH (■), and IH (▲) trials. Insulation was higher ($P < 0.05$) during IH than HH, but not during Eu.
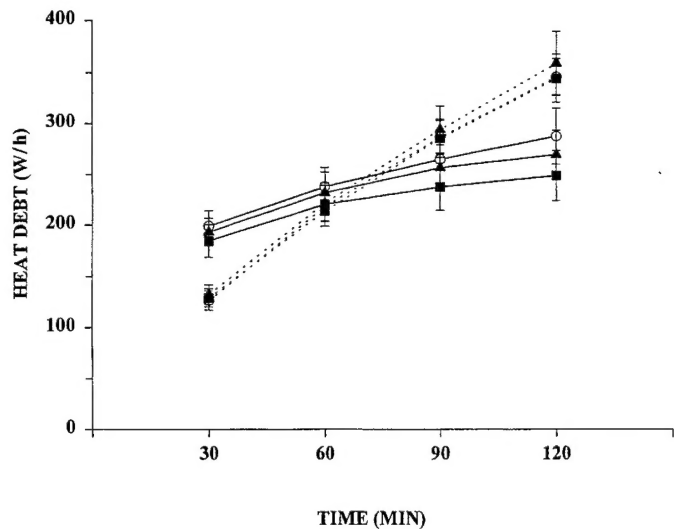
heat loss and further declines in body core temperature.

The hypohydration level studied in these experiments averaged 6–7% reduction in TBW. After hypertonic dehydration, plasma osmolality increased by ~12 mosmol/kgH$_2$O, but plasma volume did not change significantly. After isotonic dehydration, plasma volume decreased 18%; plasma osmolality increased, but only by ~5 mosmol/kgH$_2$O. Thus the three experimental conditions in this study represented normovolemic, normotonic (Eu); normovolemic, hypertonic (HH); and hypovolemic, slight hypertonic (IH) conditions.

Heat debt was similar between trials, whether determined by using the thermometric or calorimetric method. Thermometric calculation of heat debt is based on changes in body temperature, whereas partitional calorimetry balances heat loss against heat gain. Alterations in T_{sk} caused by hypohydration would therefore be expected to affect thermometric heat debt, whereas alterations in shivering thermogenesis would affect calorimetric heat debt. The lack of difference between trials in either method of measuring heat debt suggests that moderate hypohydration does not alter heat balance importantly during cold exposure.

Our findings do suggest, however, that hypohydration might impair the vasoconstrictor response to cold. During Eu, $\overline{T}_{sk}$ and T_{es}-$\overline{T}_{sk}$ gradient decreased throughout the entire cold exposure, whereas these measurements remained stable over the final 30 min of cold exposure during both hypohydration trials. The change in T_{sk} from preexposure to the end of cold exposure was ~0.6°C greater ($P < 0.05$) during Eu (−9.9 ± 0.4°C) than in either HH (−9.2 ± 0.5°C) or IH (−9.3 ± 0.3°C). Also, T_{fa}-T_f gradient tended ($P = 0.06$) to be greater during Eu than during either hypohydration trial. These observations are consistent with a weaker vasoconstrictor response to cold being elicited during the

hypohydration trials than in the Eu trial. Hence body heat may not be as well conserved during hypohydration compared with Eu.

There is some evidence in our data that the mechanism for the reduced vasoconstrictor response may be mediated by hypertonicity. T_{sk} remained higher at the end of cold exposure during HH ($23.5 \pm 0.3°C$), compared with Eu ($22.6 \pm 0.4°C$) or IH ($22.9 \pm 0.3°C$). Furthermore, insulation (an index of core-to-skin heat transfer) was lower during cold exposure during HH compared with IH. An impaired vasoconstrictor response could eventually compromise conservation of body heat and facilitate development of hypothermia, although under the moderate cold conditions employed in this study, neither heat debt nor shivering thermogenesis was affected by either type of hypohydration.

Previous research has indicated that hypohydration impairs the CIVD response (14, 15). Such effects were not apparent in our data. However, the CIVD responses in our experiment were small and inconsistent. The approach used by others to demonstrate hydration effects on CIVD involved cold exposures limited to just the hand, in contrast with our experiments involving whole body cold exposure. Recent data from our laboratory demonstrate that the CIVD response is blunted when core temperature is reduced (9). If so, hypohydration effects on CIVD may have been masked in our experiments involving whole body cold exposure.

This study is the first to examine the effects of hypohydration on thermoregulation during whole body cold exposure. The climatic conditions chosen for these experiments were anticipated to elicit shivering and peripheral vasoconstriction, as well as to reduce T_{sk} and core temperatures, but also to allow the subjects to complete the 2-h exposures. The cold stress was sufficient to cause an increase in M ($\sim 3 \times$ resting rate) and decrease in core temperature ($\sim 0.3°C$) during the 2-h exposures. Our findings provide some evidence that hypohydration may reduce vasomotor tone during cold exposure, perhaps through actions of hypertonicity on vasoconstrictor responses to cold. Thus, although hypohydration did not accelerate the decline in core temperature in these experiments, heat balance and body temperatures might be impaired by hypohydration during longer, more severe cold exposures than we studied.

REFERENCES

1. **Bittel, J. H. M.** Heat debt as an index for cold adaptation in men. *J. Appl. Physiol.* 62: 1627–1634, 1987.
2. **Boulant, J. A.** Hypothalamic mechanisms in thermoregulation. *Federation Proc.* 40: 2843–2850, 1981.
3. **Costill, D. L.** Sweating: its composition and effects on body fluids. *Ann. NY Acad. Sci.* 301: 160–174, 1977.
4. **Dill, D. B., and D. L. Costill.** Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J. Appl. Physiol.* 37: 247–248, 1974.
5. **Forgey, W. W.** (Editor). *Wilderness Medical Society Practice Guidelines for Wilderness Emergency Care.* Merrillville, IN: ICS Books, 1995, p. 25–26.
6. **Freund, B. J., and M. N. Sawka.** Influence of cold stress on human fluid balance. In: *Nutrient Requirements for Work in Cold and High Altitudes,* edited by B. M. Marriott. Washington, DC: Natl. Acad. Sci., 1995, p. 161–180.
7. **Friedl, K. E., J. P. De Luca, L. J. Marchitelli, and J. A. Vogel.** Reliability of body-fat estimations from a four-compartment model by using density, body water, and bone mineral measurements. *Am. J. Clin. Nutr.* 55: 764–770, 1992.
8. **Goldman, R. F., and E. R. Buskirk.** Body volume measurement by underwater weighing: description of a method. In: *Techniques for Measuring Body Composition,* edited by J. Brozek and A. Henschel. Washington, DC: Natl. Acad. Sci., 1961, p. 78–89.
9. **Lee, D. T., A. J. Young, J. E. Bogart, and K. B. Pandolf.** Finger vasodilatory responses in 4°C water (Abstract). *FASEB J.* 10: A116, 1996.
10. **Lennquist, S.** Cold-induced diuresis. *Scand. J. Urol. Nephrol.* 9 Suppl.: 7–46, 1972.
11. **Lewis, T.** Observations upon the reactions of the vessels of the human skin to cold. *Heart* 15: 177–208, 1930.
12. **Montain, S. J., W. A. Latzka, and M. N. Sawka.** Control of thermoregulatory sweating is altered by hydration level and exercise intensity. *J. Appl. Physiol.* 79: 1434–1439, 1995.
13. **Nishi, Y., and A. P. Gagge.** Direct evaluation of convective heat transfer coefficient by naphthalene sublimation. *J. Appl. Physiol.* 29: 830–838, 1970.
14. **Roberts, D. E., and J. J. Berberich.** The role of hydration on peripheral response to cold. *Milit. Med.* 153: 605–608, 1988.
15. **Roberts, D. E., J. F. Patton, J. W. Pennycook, M. J. Jacey, D. V. Tappan, P. Gray, and E. Heyder.** *Effects of Restricted Water Intake on Performance in a Cold Environment.* Natick, MA: US Army Research Institute of Environmental Medicine, 1984, p. 1–19. (Tech. Rept. T2–84)
16. **Sawka, M. N.** Physiological consequences of hypohydration: exercise performance and thermoregulation. *Med. Sci. Sports Exerc.* 24: 657–670, 1992.
17. **Sawka, M. N., and K. B. Pandolf.** Effects of body water loss on physiological function and exercise performance. In: *Fluid Homeostasis During Exercise,* edited by C. V. Gisolfi and D. R. Lamb. Carmel, IN: Benchmark, 1990, p. 1–38.
18. **Sawka, M. N., and C. B. Wenger.** Physiological responses to acute exercise-heat stress. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes,* edited by K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez. Indianapolis, IN: Benchmark, 1988, p. 97–151.
19. **Sawka, M. N., A. J. Young, K. B. Pandolf, R. C. Dennis, and C. R. Valeri.** Erythrocyte, plasma, and blood volume of healthy young men. *Med. Sci. Sports Exerc.* 24: 447–453, 1992.
20. **Siri, W. E.** Body composition from fluid spaces and density. In: *Techniques for Measuring Body Composition,* edited by J. Brozek and A. Henschel. Washington, DC: Natl. Acad. Sci., 1961, p. 223–244.
21. **Vallerand, A. L., G. Savourey, and J. H. M. Bittel.** Determination of heat debt in the cold: partitional calorimetry vs. conventional methods. *J. Appl. Physiol.* 72: 1380–1385, 1992.